Analytical and Clinical Evaluation of Creatine Kinase MB Mass Assay by IMx: Comparison with MB Isoenzyme Activity and Serum Myoglobin for Early Diagnosis of Myocardial Infarction

Marjan Van Blerk, Viviane Maes, Luc Huyghens, Marie-Paule Derde, Rudy Meert, and Frans K. Goris

We analytically and clinically evaluated Abbott's IMx assay for creatine kinase (CK) isoenzyme MB (CK-MB) in serum. Over a 1-year period, the method was more specific but less precise than catalytic isoenzyme measurements by electrophoresis or immunoinhibition. Sera from different individuals without electrophoretic evidence of CK-MB but containing macro CK type 1 (n = 20), mitochondrial CK (n = 5), or CK-BB (n = 5) were scored as CK-MB negative by the IMx. Likewise, CK-MB-negative by the sera remained so after addition of purified human CK-MM (≤ 7600 U/L) or CK-BB (≤ 8100 U/L). For 39 patients admitted for suspicion of uncomplicated acute myocardial infarction (precordial pain for ≤ 4 h), the diagnostic performance of the IMx CK-MB assay on admission and 4 h later was superior to that of total CK activity and compared well with that of CK-MB activity measured by electrophoresis or immunoinhibition. An admission, myoglobin showed a higher diagnostic sensitivity, specificity, and predictive value than did CK-MB and was the most informative test. Diagnostic performance on admission and 4 h later was further improved by considering positivity for myoglobin and for CK-MB by IMx and for the change in each over the first 4 h of hospitalization as criteria. Twelve hours after admission, diagnostic performance was further improved for all CK and CK-MB methods but began to decline for myoglobin.

Additional Keyphrases: heart disease · angina pectoris · agarose electrophoresis · immunoinhibition · microparticle capture enzyme immunoassay · catalytic vs immunoreactivity of enzymes

The diagnosis of acute myocardial infarction (AMI) is generally based on characteristic clinical findings and changes in electrocardiograms and serum enzymes (1–3). Measurement of creatine kinase (CK; EC 2.7.3.2) isoenzyme MB (CK-MB) in serum has long been recognized as the cornerstone of biochemical diagnosis of AMI (2). In recent years use of thrombolytic agents has increased because they are effective in minimizing myocardial damage when given within the first few hours after the onset of ischemia (4). However, such intervention requires close monitoring of CK-MB and other biochemical markers to help assess effectiveness of repermeabilization and extent of acute reperfusion damage (4). The decision to attempt thrombolytic therapy cannot be based on CK-MB measurement because CK-MB peaks too late. An earlier marker of AMI, myoglobin, has been proposed for this (5–9).

To take full advantage of the increase in biological markers during the first hours after onset of symptoms, clinicians need rapid, specific, and sensitive methods. Immunoassays of CK-MB have been developed (10–14) and are gradually being automated (15–17). Immunochemical and immunoturbidimetric assays for myoglobin allow for stat analysis of myoglobin in serum (18–20).

Here we investigate the analytical performance of a recently developed two-site immunoassay, the Abbott (Abbott, Abbott Park, IL) microparticle capture enzyme immunoassay (MEIA) method (17), compared with two other CK-MB assays based on catalytic activity measurements: agarose electrophoresis of CK isoenzymes in Paragon gels (Beckman Instruments, Brea, CA) and CK-MB Ektachem slides based on immunoinhibition (Eastman Kodak, Rochester, NY). We compared the diagnostic performance of the three assays and of a nephelometric myoglobin assay (NA Latex Myoglobin Test; Behringwerke, Marburg, FRG) in a group of patients presenting with clinical signs suggestive of AMI.

Materials and Methods

Patients

This study included 39 patients, ages 35–70 years, admitted with precordial pain (of ≤ 4 h duration) to the emergency department of our hospital. A retrospective diagnosis of AMI was made according to World Health Organization criteria for AMI and did not include myoglobin test results (21). In 17 of 20 patients with AMI, the diagnosis could be made only on the basis of clinical findings and electrocardiographic recordings. In the remaining three cases, serial enzyme measurements were also necessary. Patients with cardiogenic shock, cardiac arrest, or electrical defibrillation were excluded from the study. The patients were investigated clinically, electrocardiographically, and biochemically. Blood was sampled on admission and 4 h later. For all patients with AMI and 11 patients without AMI, serum was also sampled 12 h after admission. Fifteen patients with AMI and no patient without AMI received reperfusion therapy within the first 4 h of hospitalization. Only one patient (with AMI) had received intramuscular injections on admission, before blood sampling. At

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4 Nonstandard abbreviations: AMI, acute myocardial infarction; CK, creatine kinase; CK-MB, CK isoenzyme MB; MEIA, microparticle capture enzyme immunoassay; PPV, positive predictive value; and NPV, negative predictive value.

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that time, the concentration of myoglobin was slightly increased in this patient (105 μg/L), but CK-MB mass by IMx was borderline (5 μg/L).

Analytical Methods

**Catalytic CK (isoenzyme) measurements.** Total CK and CK-MB activity after immunoinhibition were measured at 37 °C with an Ektachem 700 XR analyzer, with use of Ektachem CK and CK-MB slides (Eastman Kodak), respectively. Fractionation of CK isoenzymes (CK-MM, CK-MB, CK-BB, mitochondrial CK, and macro CK type 1) was carried out by agarose-gel electrophoresis with fluorescence detection and quantification of CK activity (Paragon Electrophoresis System; Beckman Instruments) (11). Sera with total CK activity >450 U/L were diluted before electrophoresis. After drying, the gels were scanned with an Electrophoresis Data Center Densitometer (Helena Labs., Beaumont, TX; fluorescence mode).

**CK-MB mass determination by IMx.** The CK-MB assay on the Abbott IMx immunoassay analyzer is based on MEIA technology (22). This two-step assay uses a monoclonal anti-CK-MB antibody immobilized onto latex micro particles and a polyclonal anti-CK-MM antibody coupled to alkaline phosphatase (EC 3.1.3.1). Protein-coated microparticles are irreversibly captured by a glass fiber matrix and efficiently washed. The amount of CK-MB trapped to the beads is proportional to the bound alkaline phosphatase activity as measured by the production of fluorescent 4-methylumbelliferyl phosphate substrate (17, 22). Results are available within 40 min. The validity of the reference range advocated by the manufacturer (<5 μg/L) was confirmed by assaying sera from 82 patients (32 men, 52 women), ages 22–73 years (median 40 years).

**Myoglobin assay.** Myoglobin was determined with the Behringwerke immunonephelometric reagents consisting of shell and core particles coated with anti-myoglobin antibodies and adapted to the selective multiprotein Behring Nephelometer Analyzer. Sera were diluted 20-fold in NaCl, 9 g/L, and 13–24° forward light scatter was measured after 10 s (blank reading) and after 12-min incubation with antibody-coated latex particles.

**Sera.** Blood samples were collected and handled as previously described (11) and portions were stored at −20 °C. Total CK and CK-MB were immediately determined by immunoinhibition. The other variables were assayed within 2 weeks of sampling after frozen samples were thawed. Identification and selection of sera with pathological, atypical, or macromolecular CK forms (all from different patients) were performed according to earlier reports (23–25). In selected instances, normal sera were supplemented with purified CK-MM (from human muscle) and CK-BB (from human brain), prepared as previously described (26, 27) and kindly donated by A. Van Steirteghem (Department of Radioimmunology, Academic Hospital, Vrije Universiteit Brussel).

**Statistical Analysis**

The relation between two continuous variables was assessed by correlation analysis and by linear regression. To determine the level of significance of differences between sets of experimental data, we used the Wilcoxon signed-rank test (for paired data on activity or concentration), the Mann–Whitney U-test (for unpaired data), or the Fisher exact test (for prevalences). Stepwise linear discriminant analysis was applied to determine which combination of analytes provided the best discrimination between the AMI and non-AMI groups (28). All tests were performed two-tailed. Unless stated otherwise, P <0.05 was considered significant. To determine the clinical performance of the tested methods for AMI, we calculated their diagnostic sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) (29).

**Results**

**Analytical Evaluation**

**Precision.** Between-assay imprecision was assessed by analyzing commercial quality-control sera for 1 year (Table 1). To obtain similar numbers of observations for the different methods compared, we selected without

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Normal/decision value</th>
<th>Pathological value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>n</td>
</tr>
<tr>
<td>Total CK*, μg/L</td>
<td>127.0 (8.0)</td>
<td>40</td>
</tr>
<tr>
<td>CK-MB electrophoresis*, μg/L</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CK-MB immunoinhibition*; U/L</td>
<td>20.7 (0.7)</td>
<td>40</td>
</tr>
<tr>
<td>CK-MB MEIA*, μg/L</td>
<td>4.8 (0.7)</td>
<td>34</td>
</tr>
<tr>
<td>Myoglobin*, μg/L</td>
<td>101.0 (4.0)</td>
<td>21</td>
</tr>
</tbody>
</table>

* CK-MB control (Behring).
* CK isostrol (Sigma Diagnostics, St. Louis, MO).
* Kodak Ektachem Isoenzyme Control (Eastman Kodak).
* IMx CK-MB control (Abbott; three concentrations).
* Myoglobin control (Behring).

Table 1. Between-Day Imprecision over a One-Year Period

After omission of the correction factor determined by one-point calibration, CV=14.0% at normal CK-MB concentrations, 11.5% at moderately increased concentrations, and 9.1% at greatly increased concentrations.
Table 2. Imprecision Study for CK-MB (µg/L) by IMx

<table>
<thead>
<tr>
<th>Type of Imprecision</th>
<th>Low CK-MB</th>
<th>Medium CK-MB</th>
<th>High CK-MB</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>n</td>
<td>CV, %</td>
</tr>
<tr>
<td>Within run</td>
<td>5.7 (0.3)</td>
<td>15</td>
<td>4.6</td>
</tr>
<tr>
<td>Between run</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1 lot; 1 calibration)</td>
<td>4.2 (0.3)</td>
<td>6</td>
<td>7.1</td>
</tr>
<tr>
<td>Between run</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(1 lot; 2 calibrations)</td>
<td>4.2 (0.3)</td>
<td>14</td>
<td>7.3</td>
</tr>
<tr>
<td>Between run</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4 lots; 5 calibrations)</td>
<td>4.8 (0.7)</td>
<td>42</td>
<td>14.4</td>
</tr>
</tbody>
</table>

Fig. 1. Linear-regression analysis of CK-MB values observed with MEIA by IMx in fourfold-diluted samples compared with expected values.

Conscious bias 40 quality-control results for total CK and CK-MB by electrophoresis or immunoinhibition from the daily values for these techniques over the study period. Precision was satisfactory for catalytic CK measurements (total or CK-MB; CV mostly <7.5%) and especially for myoglobin (CV invariably <5%). CK-MB determination by MEIA was less precise (CV >10%). Omission of the correction factor, determined by the obligatory one-point calibration before each run, did not adversely affect between-run imprecision and, on the contrary, slightly improved CV at all three CK-MB concentrations tested (Table 1). Table 2 compares within-run imprecision with between-run imprecision for CK-MB by IMx with the three different CK-MB control concentrations. Between-run variability was more affected by changes in reagent lot and by recalibration at the low or medium concentration than at the high concentration. For the high concentration, between-run imprecision was much larger than within-run imprecision and was less influenced by reagent-lot changes and calibration. For CK-MB by electrophoresis, immunoinhibition, and MEIA by IMx, between-day imprecision was also compared short-term for 2 weeks by using the same two patients' sera throughout and was, respectively, 8.5%, 8.2%, and 8.4% for serum 1 (20.9 ± 1.7 µg/L by IMx; n = 10) and 7.3%, 4.9%, and 8.1% for serum 2 (80.8 ± 6.6 by IMx; n = 10).

Consistency. For the control sera in Table 1, the mean values differed by <3% from the indicated target values for myoglobin or CK-MB by IMx (except for the lowest concentration: target value = 5.0 µg/L; experimental mean = 4.6 µg/L). Addition of calibrator to patients' sera yielded recoveries of 85-96% for CK-MB by IMx and for myoglobin (n = 2-4).

Linearity. To evaluate assay linearity, we analyzed samples with increased CK-MB concentrations diluted with the zero calibrator from the Abbott kit, and samples with increased myoglobin concentrations diluted with NaCl, 9 g/L. For myoglobin, linearity was excellent at least up to 350 µg/L (r = 0.998, S_50 = 0.3 µg/L for the dilution series by linear-regression analysis). Linearity was not, however, achieved for the Abbott kit, because diluted sera systematically yielded higher results. This problem was largely overcome by diluting the samples in fresh CK-MB-negative human serum (containing <2 µg/L CK-MB; results for diluted samples were corrected for the actual low amount of CK-MB mass). Figure 1 compares expected (i.e., when assayed undiluted) and measured CK-MB concentrations for 14 samples (observed range when undiluted: 48-234 µg/L; linearity range stated as <300 µg/L) after fourfold dilution with either zero calibrator or CK-MB-negative serum. Dilution in zero calibrator on average yielded 19% higher results (P <0.001) than did undiluted serum. By contrast, dilution with CK-MB-negative serum produced no significant difference from undiluted sera (only 4% difference between the mean values of each group).

Correlation. As illustrated in Table 3, CK-MB values obtained by different methods were significantly correlated, as were the results for samples requiring dilution. When MEIA results were compared with results from another method, the slope increased for samples diluted in zero calibrator. As could be expected, the various methods yielded different results, illustrated by slopes dissimilar from 1 and by the presence of an intercept (of ~10 U/L) for comparisons involving Ektachem slides.

Specificity. Serum samples containing macro CK type 1 (11-202 U/L; n = 20), mitochondrial CK (6-39 U/L; n = 5), or CK-BB (5-61 U/L; n = 5) were selected on the basis of typical electrophoretic patterns and high-performance gel-permeation chromatography (23-25). In all cases, CK-MB concentrations measured by the IMx fell within the reference limits (<5 µg/L), whereas immunoinhibition invariably yielded higher values. Addition of large amounts of purified human CK-MM (<7600 U/L) and CK-BB (<8100 U/L) to a CK-MB-negative serum sample did not induce CK-MB positivity as judged by the Abbott kit.
Clinical Evaluation

We next investigated the diagnostic performance of the three CK-MB assays and measured total CK and myoglobin in 39 patients admitted to the emergency department with typical clinical findings suggestive of AMI. According to clinical, electrocardiographic, and biological data, 20 patients were retrospectively classified as having an AMI (19 acute, 1 patchy necrosis). The non-AMI group comprised 19 patients, with the following diagnoses: atrial fibrillation (n = 2), hyperventilation (n = 1), cholelithiasis (n = 1), cholecystitis (n = 1), atypical precordial pain (n = 3), and angina pectoris (n = 11). On admission, the median duration of chest pain was 120 min (range 60–240 min) in the group with AMI and 120 min (range 30–240 min) in the group without AMI (P >0.05). Median age was 58 years (range 37–70 years) in the AMI group and 56 years (range 41–68 years) in the non-AMI group (P >0.05). The AMI group consisted of 18 men and 2 women; the non-AMI group was 11 men and 8 women (P = 0.03). In the non-AMI group, concentrations of myoglobin and CK-MB (whether determined by electrophoresis, immunoinhibition, or MEIA) were not statistically different between admission and 4 h later. For total CK, a statistically significant decrease (P = 0.04) was noticed. The AMI and non-AMI groups differed only in myoglobin concentration (P = 0.03) on admission (Figure 2), but 4 h later, the differences between groups for total CK, CK-MB, and myoglobin were highly significant (P <0.001).

Stepwise discriminant analysis was applied to determine which set of variables allowed the best discrimination between the AMI and the non-AMI groups on admission. Myoglobin allowed the best possible discrimination between both groups, with 70% of cases correctly classified. The other tests did not provide any additional relevant information. Stepwise linear discriminant analysis applied to the remaining four variables (total CK and CK-MB by electrophoresis, immunoinhibition, or MEIA) again identified one variable: CK-MB, as determined by immunoinhibition, which allowed correct classification of 64% of cases.

Table 4 summarizes the diagnostic performance of the tests on admission and 4 h later. With the upper reference limits stated by the manufacturers (150 U/L...
for CK, 6% of total CK activity for CK-MB by electrophoresis, 16 U/L for CK-MB by immunoinhibition, 5 μg/L for CK-MB MEIA, and 90 μg/L for myoglobin), diagnostic sensitivity was low for all tests, ranging from 0% for electrophoresis to 30% for myoglobin, whereas specificity was highest (89–100%) for CK-MB methods (especially electrophoresis) and myoglobin on admission. A Spearman rank correlation test showed no significant correlation between duration of precordial pain on admission and myoglobin concentrations on admission for AMI patients (r = 0.334). The PPV of increased total CK activity on admission was relatively low (43%) compared with that of increased CK-MB concentrations (~70%, irrespective of method). Upon admission, PPV was highest for myoglobin (100%); NPVs were low for all methods (47–58%). For samples obtained within 4 h after admission, the diagnostic performances of CK-MB and myoglobin assays were invariably superior to that of total CK, with PPVs of 85–100% (highest for myoglobin and immunoinhibition) and NPVs of 76–84% (highest for myoglobin and CK-MB MEIA). When the change in CK, CK-MB, or myoglobin concentrations during the first 4 h after admission exceeded the 97.5 percentile (the limit established for similar changes in the non-AMI group), the sensitivity and NPV of such diagnostic criteria tended to be higher for most methods than for measurements taken only once. Joint positivity for CK-MB (e.g., by MEIA) and myoglobin generally gave very high diagnostic specificity and PPV at each time or for changes over the 4 h. Positivity for CK-MB or myoglobin tended to maximize sensitivity and NPV, especially when concentration changes over 4 h were considered. For 20 patients with AMI and 11 patients without AMI, samples were also available at 12 h after admission. For total CK and CK-MB by electrophoresis, no false positives or false negatives were noted; for CK-MB by immunoinhibition or MEIA, one false positive each (different patients) and no false negatives were noted; and for myoglobin, no false positives and four false negatives were noted. In general, PPV and NPV tended to increase for all CK and CK-MB methods at 12 h after admission, whereas NPV for myoglobin decreased.

**Discussion**

The Abbott IMx assay for CK-MB very specifically recognizes this isoenzyme as evidenced by analysis of sera selected on the basis of their electrophoretically determined isoenzyme composition and of samples supplemented with purified human CK isoenzymes. This lack of interference of CK-MM and CK-BB isoenzymes confirms the results of Brandt et al. (17) and enables CK-MB measurement without prior dilution in the presence of high total CK activity. In agreement with evaluations of similar immunoaassays (10–12, 15), the IMx assay is relatively imprecise in comparison with catalytic assays. Brandt et al. (17) and Sasse et al. (30) reported slightly better precision for CK-MB by IMx (CV ≤10% at all three concentrations tested). Their results were, however, obtained for only 1 month (vs 1 year here) and were based on concentrations calculated from one calibration curve. In such a setting we also found CVs <10%.

The imprecision of the CK-MB IMx assay may interfere with consistent detection of borderline increases of CK-MB concentrations and seems not to benefit from the obligatory one-point calibration before each run. Variability between runs, calibrations, and reagent lots all contribute to overall imprecision by IMx, albeit to a varying degree according to CK-MB concentration and the nature of the control serum used. However, because changes in CK-MB concentrations with time are generally monitored to support the diagnosis of AMI, the still improvable imprecision of the IMx assay of CK-MB is no obstacle for its clinical usefulness, as discussed later.

Our analytical evaluation of the IMx assay demonstrated nonlinearity of dilution curves obtained by diluting patients' sera with zero calibrator, which is claimed to consist of human serum. This can lead to
overestimation of CK-MB concentrations by ~20% and hence to artifactual variations in CK-MB concentrations during follow-up, depending on the degree of sample dilution. Our results disagree with those of Brandt et al. (17), who contend that dilution with zero calibrator is satisfactory. This problem can be overcome by using CK-MB-negative human serum as diluent, which indicates that changes in sample matrix may influence results.

The use of human serum instead of zero calibrator for diluent seems mandatory for quantitative treatment of serial CK-MB measurements, e.g., for evaluating the extent of myocardial damage. However, unless care is taken to select noninfectious blood-donor material, the use of human sera as diluent exposes laboratory workers to unnecessary health hazards. Therefore, we urge manufacturers to eliminate the problem by providing suitable diluents.

Nevertheless, results obtained by IMx correlated very significantly with catalytic measurements of CK-MB by electrophoresis and immunoinhibition whether or not samples requiring dilution were included, confirming the well-established concordance of CK-MB activity and mass concentrations during the early evolution of AMI (31, 32). For myoglobin, the data obtained for precision and consistency were satisfactory and compared well with the results of other authors (18, 20).

The relative analytical imprecision of the IMx assay might help explain why its diagnostic specificity for AMI was paradoxically slightly lower than the specificity of catalytic CK-MB measurements in a series of 39 patients admitted for suspicion of AMI on the basis of typical clinical findings. Although this contention is compatible with the borderline increases in CK-MB detected by IMx (but not by catalytic CK-MB methods or for myoglobin) in three patients without AMI (see Figure 2), the consistently increased CK-MB mass in one patient with crescendo angina pectoris might reflect minor necrosis in the absence (by WHO criteria) of AMI (14, 33). On the other hand, CK-MB by IMx showed higher diagnostic sensitivity, especially at onset, and better NPV than did catalytic CK-MB assays. Cutoff values proposed by the manufacturers for catalytic CK-MB assays should be used cautiously, because severe muscle damage can produce increased CK-MB concentrations of noncardiac origin. In such cases, it may be preferable to express CK-MB as a percentage of total CK (30). At lower total CK concentrations, as here, absolute values may be mandatory. Minor injuries to skeletal muscle frequently coexist with AMI, and expressing CK-MB as a percentage of total CK may adversely affect clinical efficiency of the test (10). Likewise, the ratio of CK-MB activity or mass to total CK is quite insensitive in diagnosing periperoferary myocardial infarction after coronary bypass surgery (34). Overall, the diagnostic performance of the different CK-MB assays tested was comparable also with results from similar studies (35) and, as in other reports, surpassed that of total CK determinations (36).

Myoglobin measurements proved to be more informative on admission than were CK-MB measurements, irrespective of methodology, as evidenced by stepwise discriminant analysis and overall better diagnostic performance. In agreement with Ohman et al. (9), no significant correlation existed between the duration of symptoms and the serum concentration of myoglobin of AMI patients on admission. The diagnostic specificity and predictive value of myoglobin for AMI might, however, be overestimated in this series because only uncomplicated AMI was considered here and only one patient had received intramuscular injections before blood sampling on admission. Indeed, myoglobin is inherently not cardiac specific (37) and may even be moderately increased in myocardial ischemia, indicating subclinical myocardial necrosis (6, 8, 33). The present data, dealing with a very short delay between the onset of chest pain and admission to hospital, further support the view that myoglobin is a useful adjunct of CK-MB as biological marker of AMI for early diagnosis (5-9). If hospital admission is delayed, however, myoglobin concentrations may already have fallen (5). Likewise, in our study diagnostic performance of myoglobin tended to decrease at a later time (12 h after admission), whereas diagnostic performance continued to increase for all CK and CK-MB methods. For all variables investigated, comparison of different times improved diagnostic performance, thus confirming the utility of serial determinations (5, 38). Note, however, that in most AMI patients reperfusion therapy was attempted (vs none in non-AMI patients). This intervention may have hastened the increase in CK-MB and myoglobin during the first 4 h of hospitalization in the AMI group, thereby increasing the difference in marker kinetics between groups (39).

In summary, the CK-MB assay by IMx is highly CK-MB specific but has less than perfect analytical sensitivity and linearity. Its diagnostic performance in patients with suspected AMI is comparable with current CK-MB assays and benefits from serial analysis and joint determination of myoglobin. Its specificity and independence from the specific activity makes the method particularly suitable for infarction sizing, retrospective diagnosis, and assessment of CK-MB in special instances (e.g., abnormally high residual CK-MB activity with immunoinhibition). Until IMx has random-access capability, the method is less suitable in true stat situations. Determination of myoglobin at two points during the first hours after admission appears a useful adjunct to early biochemical diagnosis of AMI. In this regard, stat analysis by the Behring Turbitimer seems an attractive alternative because this instrument yields results for myoglobin within 2 min that compare well with nephelometry by the Behring nephelometer.

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References